Correspondence

Letters to the Editor should not exceed 500 words.

Pressurized Aerosols in Asthma

SIR,—We suspect that patients with asthma may be killing themselves by the excessive use of sympathomimetic agents in the form of metered or pressurized aerosols containing isoprenaline, orciprenaline, or adrenaline. We have had four patients known to us previously as having mild or moderate asthma and who have been found unexpectedly dead at home or at work. By their side or in their hand has been an empty or almost empty pressurized aerosol. These patients have not had very severe attacks of asthma, and nothing has been found at post-mortem examination to suggest that asthma was the cause of death, nor has any other lesion been found.

In eight other patients admitted to hospital for varying degrees of severe asthma treatment has been carried out, but sudden and unexpected death has taken place without obvious cause, and little abnormal has been found at post mortem to account for this. In all there has been a marked tachycardia. In one an electrocardiogram confirmed the tachycardia with multiple ectopic beats and coupling. Either the patients or their relatives told us that they had used one or more pressurized inhalers during the preceding few days because of intractable wheezing.

It is difficult to prove that these deaths have been due to excessive doses of sympathomimetic compounds. In most of these patients adrenocortical insufficiency was unlikely, and few were on long-term steroids. Presumably their deaths were due to ventricular fibrillation, itself due to the absorption of sympathomimetic compounds. There are ample quantities of these compounds in each inhaler to cause death if the total amount is absorbed systemically in a short time through the lungs. Unfortunately so far we have not managed to secure an electrocardiogram taken at the moment of death, but in the one case quoted the ventricular tachycardia with multiple ectopic beats was probably a forerunner of ventricular fibrillation.

We feel that our evidence is strong enough to justify warning each patient using such metered or pressurized aerosols. A label should be put on each inhaler, and not merely with the instructions or on the outside box, specifying that not more than one puff should be taken and at not more than half-hourly intervals. It should be stated that it is dangerous to exceed this amount. So far as we are aware there are no such warnings given on any of the aerosol containers or with the instructions, with one or two exceptions, where the warning is not very prominent.

Treatment of asthmatic patients in whom it is believed that there has been an excess of sympathomimetic drugs is difficult. Beta-receptor antagonists such as propranolol should theoretically be of value, although their bronchoconstrictor action may make them rather dangerous.—We are, etc.,

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Ware Park Hospital, Ware, Herts. A. PINES.

Psychiatric Sequelae of Termination of Pregnancy

SIR,—Drs. D. W. K. Kay and K. Schapira appear (4 February, p. 299) to be guilty of the very "distortion" of which they accuse you. They quote extensively from the Scandinavian literature to support their contention that there are no serious sequelae to the termination of pregnancy. However, statements and statistics in the articles from which they quote can be used to support the opposite view.

For example, Jansson[1] states categorically that "symptoms of insufficiency are strikingly more uncommon after legal abortion than after other abortions, and also distinctly more common than after delivery." Again, Höök[2] found that 73% of women refused abortions were satisfied at the way things developed. The writers doubt the validity of the figure of 59%, because they have failed to discover the data from which the figure is derived. Arén and Amark[3] point out that unfavourable reactions after legal abortion are not unusual, but occur in 35% to 60% even if these reactions are rarely of a severe degree.

Most psychiatrists have heard the threat of turning to illegal abortion and suicide if the termination of pregnancy is refused. Lindberg[4] alone seems to have investigated these matters properly, and he found that those who turn to illegal abortion number 12% at the most, and there was a complete absence of suicide despite the high frequency of threats.

This shows, therefore, that it is not politicians alone who can use the same set of figures to prove the very opposite hypotheses, and, more important, proves that we are still far from having a clear picture of the true facts regarding the sequelae of termination of pregnancy; that further investigation and collation of facts are needed, followed by correct interpretation of the findings, before we can seriously consider how they should be used to improve, if not to solve, the present problem of abortion.

The call, therefore, should be for more facts, and less fiction, fantasy, and feelings. It would be of interest to know what *facts* the present members of the House of Commons Standing Committee will be using to produce a Medical Termination of Pregnancy Bill. —I am, etc.,

Shrewsbury. M. DAVID ENOCH.

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[1] Jansson, B., *Acta psychiat. scand.*, 1965, **41**, 87.
[2] Höök, K., ibid., 1963, Suppl. No. 168.
[3] Arén, P., and Amark, C., *Svenska Läk.-Tidn.*, 1957, **54**, 3709.
[4] Lindberg, B. J., ibid., 1948, **45**, 1381.

SIR,—The letter from Drs. D. W. K. Kay and K. Schapira (4 February, p. 299) criticizes your leading article (3 December 1966, p. 1342) as well as the report of the Royal College of Obstetricians and Gynaecologists and points out the factual errors. Their letter would convey that they are sitting in independent judgement of loose statements and that they are merely interested in producing the relevant facts. Ekblad,[1] who is quoted, has of course written the most comprehensive monograph on the follow-up of women (479) who had a therapeutic abortion on psychiatric grounds, but his conclusions are much wider than those which their letter implies.

Firstly, the tribunals selecting patients were feeling their way and a sizable number were selected for abortion who in the light of Ekblad's findings should not have been selected at all, for of those who had the abortion 25% expressed regret. It is likely that this represents a conservative estimate, for a woman who has persuaded a tribunal to abort her is unlikely to round on them later and say she regretted it, if only not to prejudice her chances of a second abortion. Of the 427 who were not simultaneously sterilized no fewer than 156 (37%) had become pregnant again before Ekblad's follow-up.

Drs. Kay and Schapira have minimized the psychiatric sequelae following abortion, but Ekblad's summary (p. 218) gives his views very clearly and they should be quoted:

"The psychically abnormal find it more difficult than the psychically normal to stand the stress implied in a legal abortion. This means that the greater the psychiatric indications for a legal abortion are, the greater will be the risk of unfavourable psychic sequelae."

These conclusions are very much in accord with my own,[2] and it is of interest that Ekblad[3] came to a similar conclusion in his study on sterilization.

I would agree that the mentally normal with no psychiatric indications for termination are less likely to come to psychiatric harm following an abortion, but is this the issue in the Medical Termination of Pregnancy Bill? Those who advocate abortion for the unwanted child should take a hard look at Ekblad's figures of those women who regretted their abortion. A conservative figure of 25% is a very big error and reinforces what many of us have stressed: that "not wanting" is a temporary state of mind. Ekblad's comment on the increased risk of "serious self-reproach" if the woman had been influenced by others to submit to abortion is also very relevant, for many of the unmarried are pressed to have an abortion by their parents and many of the married are strongly influenced by their husbands.

It would be helpful if those British psychiatrists who advocate abortion on psychiatric grounds would supply an analysis of their data comparable to that of Ekblad. It is a serious omission that British academic medicine in the psychiatric, gynaecological, and social medicine fields has not yet reported. We have too much propaganda but not enough information.—I am, etc.,

Queen Elizabeth Hospital, Birmingham. MYRE SIM.

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Bleeding Peptic Ulcer

SIR,—The letter from Mr. T. J. Butler (11 February, p. 360) commands attention and will carry weight.

Many of us share his opinion that reflux of duodenal secretions into the stomach may play a part in degenerative gastric disease. May I therefore suggest that he should modify the operation he suggests for bleeding gastric erosions by omitting the vagotomy. It is wise for those in the vanguard of medical research, or those producing new operative procedures, to proceed one step at a time. The first thing to prove in the interests of our patients is that a Roux-en-Y anastomosis following gastric resection will stop the bleeding from gastric erosions. If vagotomy is added on theoretical grounds it will have two disadvantages. The first is that it increases the severity of the operation, particularly when selective vagotomy is used, and so increases the mortality for operations for bleeding gastric erosions. Secondly, we shall still not be certain whether it is the Roux conversion or the vagotomy which has had the beneficial effect.

May I suggest, therefore, that a more logical step for Mr. Butler and those who follow him would be simply to carry out hemigastrectomy completed by Roux-en-Y anastomosis. The patients should then be carefully followed up to confirm whether in fact the gastric acidity rises significantly. Needless to say, a sympathetic attitude to any complaints made by the patient must thereafter be maintained in order to anticipate the onset of jejunal ulceration and to deal with it at once by total or "gastric" vagotomy, as favoured by the surgeon.—I am, etc.,

London W.1. NORMAN TANNER.

Fucidic Acid for Staphylococcal Infections in Children

SIR,—The object of this communication is to report clinical experience with fucidic acid in the oral treatment of acute staphylococcal infections of the skin and lung in children. Previous reports in adults suggest that fucidic acid is a potent antistaphylococcal agent, but experience with children is lacking.[1-12]

Seven children aged 14 months to 4 years were referred to our hospital suffering from widespread staphylococcal skin infection and fever. Three of these patients had previously received sulphonamides for seven to ten days, and two penicillin for seven days without effect. Each patient was given 30 mg. of fucidic acid per kg. body weight per day for seven to ten days. Three patients aged three, six, and two months respectively were referred with dyspnoea and fever. Chest x-rays were characteristic of staphylococcal pneumonia. Two of them had been treated elsewhere previously, one with penicillin and methicillin and the other with penicillin, chloramphenicol, and cloxacillin with no effect. The two-month-old patient did not receive any treatment. These three patients were given 30 mg. of fucidic acid per kg. body weight per day for 20 days. In the third patient penicillin was also given intramuscularly.

Swabs from the skin or the pharynx revealed in all ten patients coagulase-positive staphylococcus resistant to penicillin, streptomycin, and tetracyclines, but sensitive to erythromycin, methicillin, novobiocin, and fusidic acid. In staphylococcal skin infection the lesions cleared completely within five to seven days, and temperature returned to normal within three to four days of treatment.

Two of the patients with staphylococcal pneumonia improved remarkably five to seven days after the institution of fucidic acid therapy. Temperatures dropped to normal, dyspnoea became much less pronounced, and the patients began to gain weight. In the first patient chest x-rays were normal within 15 days of treatment. In the second patient there was considerable radiological improvement, but a small bulla in the right lower lobe persisted 20 days after the beginning of treatment. It disappeared two months later without any additional treatment. The third patient became apyrexial and less dyspnoeic within four days and chest x-rays completely cleared 16 days after the treatment with fucidin.

No side-effects were observed in any of the patients.—We are, etc.,

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Accidental Hypothermia

SIR,—The report of the Committee on Accidental Hypothermia of the Royal College of Physicians raises once again the problem of safe rewarming rate following accidental hypothermia (17 December, p. 1471).

From a study of 71 episodes of prolonged induced hypothermia (30–198 hours at 29–33° C.) conducted in conscious subjects at the Royal Marsden Hospital, London, during 1960–3,[1] it would seem that the only safe or "ideal" rate of warming following accidental hypothermia is that which does not give rise to complications during rewarming. This will differ from patient to patient, depending on duration and depth of hypothermia, coincident disease, and on rise in blood volume, renal water excretion, and cardiac output during rewarming. A rise in rectal temperature of 0.5° C. per hour may serve as a useful guide to rewarming rate when duration of hypothermia has been comparatively brief but may not be safe following more prolonged hypothermia. The rate of rise of rectal temperature during spontaneous rewarming may vary from 1° C. in 60 minutes following 30 hours at 30° C., to 1° C. in 400 minutes following 192 hours (8 days) at 30° C. There does not appear to be any harm resulting from slow rewarming following prolonged hypothermia. Active rewarming in this situation should be reserved for those patients in whom spontaneous rise in body temperature does not occur. In these subjects it is probably wise to limit gentle active rewarming (a heat blanket placed over a cotton or wool blanket) to not more than half an hour at a time.

In the above study patients were mostly conscious and rational, with stable blood pressure, heart rate, and respiratory rate, during controlled hypothermia. This made it possible to observe rewarming from a known baseline and to distinguish clinical changes resulting from rewarming from those due to hypothermia and/or to coincident disease. Rewarming changes in these subjects included evidence of cerebral compression (paresis, papilloedema, increase in lumbar cerebrospinal-fluid pressure, confusion, coma), acute pulmonary oedema (neurogenic or circulatory in origin, and liable to be mistaken for fulminating bronchopneumonia), central circulatory failure, gastric dilatation, gastric retention (with the risk of vomiting and aspiration of vomitus), and mucosal haemorrhage. It seems probable that these complications result from increase in intravascular and interstitial fluid volume, and in intracranial pressure.

If rewarming following accidental hypothermia is analogous to that following prolonged induced hypothermia, the following measures might be beneficial in the management of this condition:

(1) Lumbar cerebrospinal-fluid pressure should be checked daily and lumbar puncture repeated as frequently as is necessary to control pressure rise. Intracranial pressure may rise to 500–600 mm. cerebrospinal fluid during rewarming, and can be reduced by 200 mm. or more without risk, with dramatic relief of cerebral compression or neurogenic pulmonary oedema.

(2) A rapidly acting diuretic might usefully be given if there is evidence of cardiac failure or of pre-existing heart disease, or if renal water excretion has not risen substantially by the time rectal temperature rises to 35°–37° C. Sweating is largely absent during rewarming and increase in blood volume is compensated by renal water excretion.

(3) Regular aspiration of gastric contents. If this is normal in colour, volume, and consistency it should be returned to the stomach.

(4) Avoid intravenous transfusion.

(5) Patients should be covered by a sheet or one or two blankets to prevent shivering (and according to comfort), and rewarming should be spontaneous. The high mortality associated with active rewarming following *prolonged* hypothermia has been recognized for more than 25 years. Occasionally even spontaneous rewarming may be unduly rapid, leading to severe clinical deterioration. If this occurs and cannot be controlled by the above measures, the patient should be cooled by 2–4° C. (using pethidine, chlorpromazine, and surface cooling) and body temperature stabilized for 4–6 hours before permitting further rise in temperature.

The changes of rewarming take several days to resolve and close observation of